Stenting for Abrupt Closure of the Intracranial Vertebral Artery Complicating Balloon Angioplasty

A Case Report

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Key words: stent, intracranial vertebral artery, acute occlusion

Summary

We report a case of stenting for abrupt closure of the intracranial vertebral artery complicating balloon angioplasty. A 58-year-old man with symptomatic restenosis of the intracranial vertebral artery underwent balloon angioplasty, which was complicated by acute occlusion due to wall dissection. The acute occlusion of the lesion was completely recanalized by implanting a balloon-expandable stent designed for the coronary artery. Follow-up angiography 15 months after stenting did not show severe restenosis and the patient's symptoms disappeared after stenting. This therapeutic option may be useful as a means to bail out from acute occlusion of the intracranial artery caused by endovascular procedures.

Introduction

Percutaneous transluminal angioplasty (PTA) has evolved into a viable treatment alternative not only for stenoses of the extracranial arteries but also for stenoses of the intracranial arteries. PTA for cerebrovascular lesions, however, sometimes induces fatal complications such as acute occlusion due to wall dissection [7,11]. In the coronary artery, stents have been used to treat dissections or acute closure after percutaneous transluminal coronary angioplasty (PTCA) [5,6]. Herein we describe an instance of the successful use of a balloon-expandable stent designed for the coronary artery to treat acute occlusion due to wall dissection of the intracranial vertebral artery caused by the PTA procedure.

Case Report

A 58-year-old man with a medical history of diabetes mellitus and hypertension experienced multiple episodes of dizziness and drop attacks over a six month period. MRI showed an infarction in the right occipital lobe. Angiography showed complete atherosclerotic occlusion of the left vertebral artery at the V1 portion and severe stenosis involving the right intracranial vertebral artery (figure 1A).

Atherosclerotic change without severe stenosis was shown in the other craniocervical arteries, and collateral flow from anterior circulation to posterior circulation was poor. PTA for stenosis of the right vertebral artery was performed since ticlopidine did not improve the ischemic symptoms.

Although angiographical wall dissection of the vertebral artery was detected, we obtained

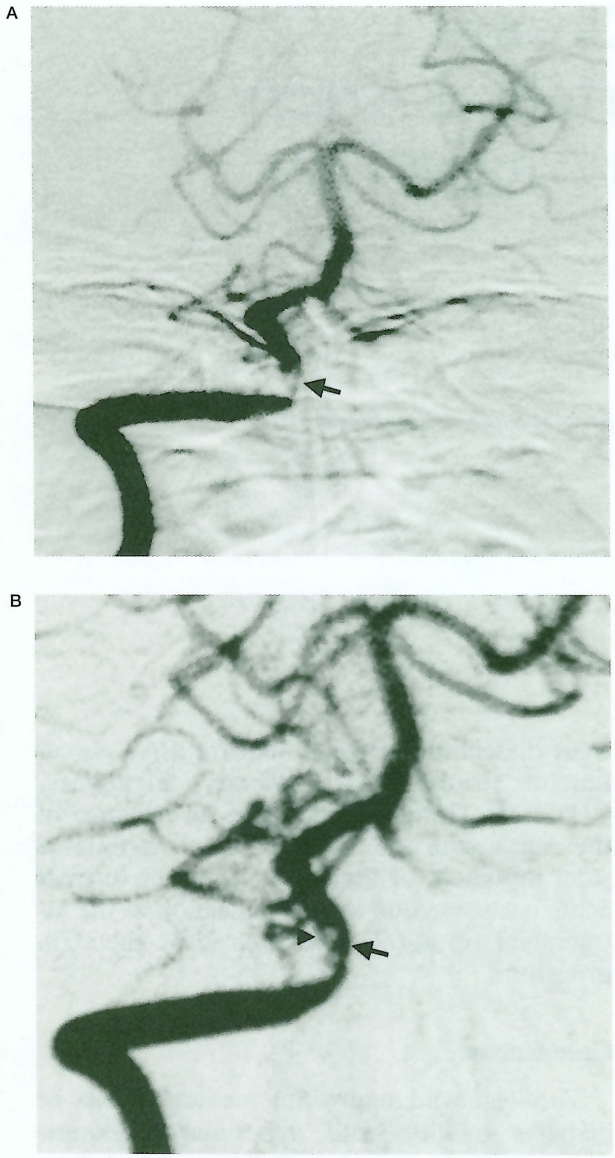

Figure 1 A) Anteroposterior view of right vertebral angiography of a 58-year-old man experiencing drop attack and dizziness showing severe stenosis of the right intracranial vertebral artery (arrow). B) Vertebral angiography immediately after first PTA revealing dilatation of the lesion (arrow) and wall dissection (arrowhead).

dilatation of the lesion (figure 1B). After the PTA, his symptoms disappeared. However, dizziness recurred frequently five months after PTA. Follow-up angiography six months after PTA showed severe restenosis (figure 2A). We performed PTA for the lesion again.

The endovascular technique was performed from a transfemoral approach under local anesthesia. A sheath (6 French) was inserted into the right femoral artery, and systemic anticoagulation was achieved by administering intravenous heparin and determining the activated clotting time (2.0 times the baseline value). A guiding catheter (100 cm in length, 6 F in diameter) was positioned in the proximal right vertebral artery. A 0.014-in guidewire was navigated across the stenosis. The balloon catheter (2.0 mm in diameter, 1.5 cm in length) was then guided over the wire, and inflated to the balloon pressure of 3 atm for 30 seconds. Fluoroscopy revealed poor dilatation of the lesion. Secondary dilatation was performed with a larger balloon catheter (2.5 mm in diameter), which was used in the previous PTA procedure, for 30 seconds. After the balloon was deflated, the patient moved strongly and vomited. His consciousness was acutely disturbed to a comatose state. Angiography showed occlusion of the vertebral artery at the lesion (figure 2B). However, the guidewire was positioned beyond the lesion and it was able to pass through the true lumen of the lesion with the tip positioned in the posterior cerebral artery to ensure maximal wire support. A balloon-expandable stent (gfx 2.5 mm in diameter, 12 mm in length) was deployed over the wire at the occlusion site (figure 2C).

Final angiography showed sufficient dilatation of the lesion (figure 3D). Consciousness disturbance was resolved after recanalization of the lesion. After the stenting, symptoms disappeared. Follow-up angiographies were performed at three, six and 15 months after stenting and showed good patency of the lesion with mild restenosis.

Discussion

Stenotic lesions of intracranial arteries have recently become approachable by endovascular techniques. Several reports have described successful results of PTA for intracranial vertebral arteries [7,11]. However, PTA has some potential complications including vessel dissection, acute occlusion, and thromboembolism from disruption of atheromatous plaque and the dissection site. In this case, we thought the cause of acute occlusion was wall dissection, because dissection was detected on the angiography just after previous balloon angioplasty. If wall dissection and abrupt closure occurred, we had to perform further angioplasty until successful dilata-

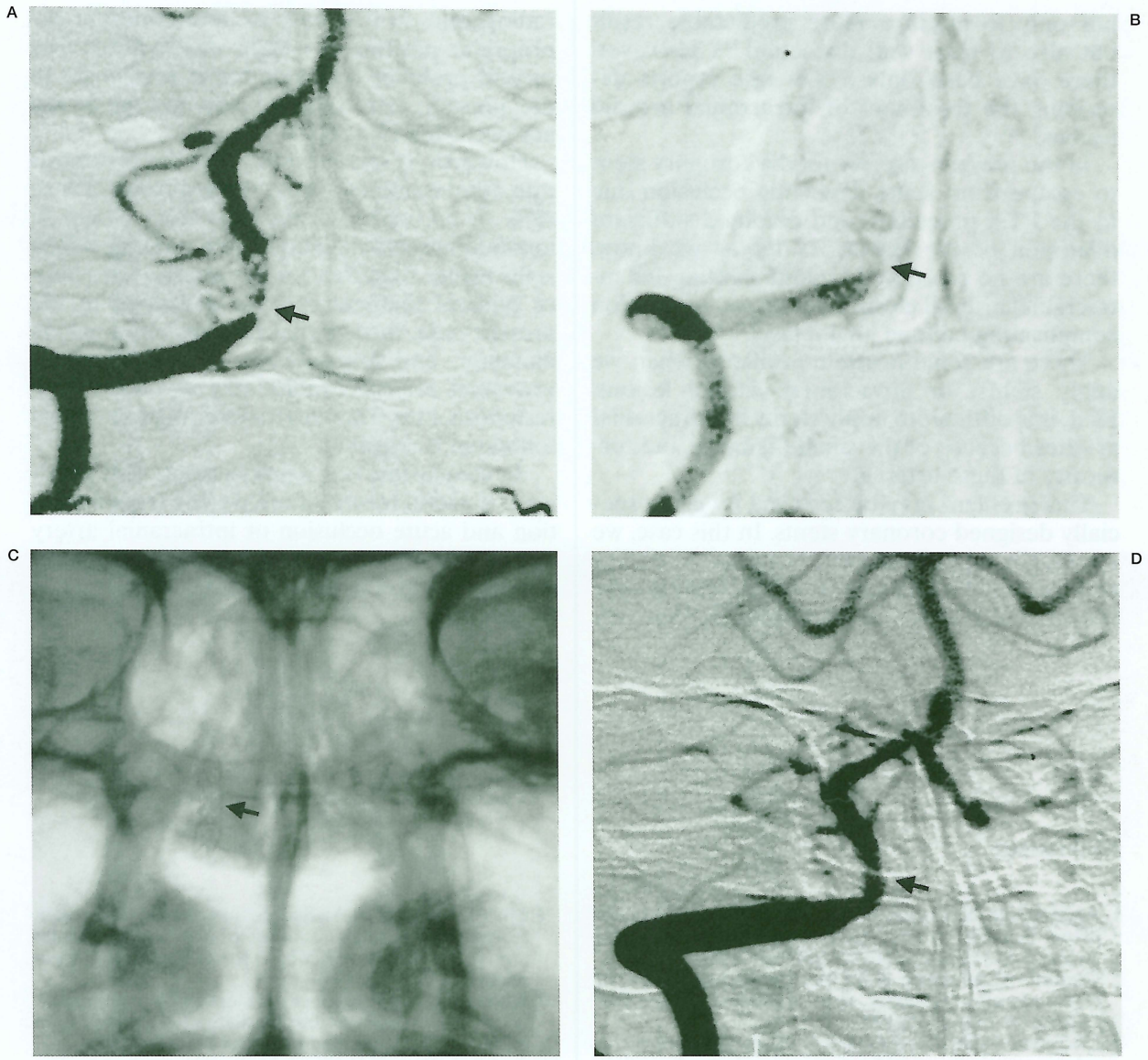

Figure 2 A) Vertebral angiography 6 months after first PTA showing severe restenosis of the vertebral artery at previously treated segment (arrow). B) Angiography just after balloon angioplasty showing occlusion of the vertebral artery at the angioplasty site (arrow). C) Anteroposterior radiograph after stenting showing the 2.5 mm expanded stent (arrow). D) Angiography just after stent implantation showing sufficient dilatation of the lesion (arrow).

tion was confirmed to prevent ischemic complications. It is almost impossible to recover completely from occlusion due to wall dissection only by balloon angioplasty. In the presented case, however, the passage of the dissecting segment with the guide wire was thought to be hazardous; we thought immediate recanalization of the lesion had to be performed, because conciousness disturbance of the patient acutely progressed.

So we performed stent placement, which was thought to be the most certain method to recanalize the occlusion site caused by wall dissection immediately. Regarding the coronary artery, stenting was used as a salvage method for significant dissection or recoil after failed PTCA and its success rate was over 95% [5,6]. Stent placement has recently been reported in the treatment of lesions of the craniocervical artery, especially in the internal carotid artery,

not only to attain a more predictable result but also to treat wall dissection [3,12]. However, there have been only a few case reports describing the treatment of intracranial arterial lesions [1,3,4,9,10].

In the present case, we used a coronary stent to escape from acute dissecting occlusion due to the PTA procedure and obtained sufficient dilatation of the lesion. As far as we know, there are no reports of bailout stenting for intracranial arterial closure due to wall dissection complicating balloon angioplasty.

There are some possible problems when we apply stents for intracranial arterial lesions. First, it is difficult to apply stents in intracranial arteries because of the small diameter and tortuosity of these arteries.

This problem may be resolved by using specially designed coronary stents. In this case, we used a gfx stent. Because it has good flexibility and trackabilty, it can be tracked well into the intracranial arteries. In addition, it is relatively radiopaque, so it is easy to determine the appropriate position for stent deployment. The second problem with stenting is restenosis. In the present case, symptomatic restenosis did not occur for 15 months.

However, it was reported that the restenosis rate after stent placement ranged from 23% to 48% in coronary arteries less than 3 mm in minimal luminal diameter after stenting, which is far higher than that for the arteries with larger luminal diameter [2,8,13]. As described in the present case, intracranial arteries are small in diameter, and implantation of stents for intracranial arterial lesions may involve a high restenosis rate. Therefore, close angiographical follow-up is required.

In conclusion, our results suggest that stenting is a useful means of recovering from dissection and acute occlusion of intracranial artery due to PTA. We recommend that PTA for cerebrovascular lesions be performed with backup by stenting.

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